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This contract supported two investigations of the effects of alcohol on humans. The primary project was the investigation of the effects of alcohol on information processing and memory. The second project was the phase-out of our studies of the effects of acute and chronic alcohol ingestion on physiological sleep patterns.

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The long-term goal of this research has been to increase our knowledge of the effects of alcohol on human performance. The long-term expectation is that analysis of the effects of alcohol on information processing tasks will reveal the locus of alcohol effects on the different hypothetical cognitive processes involved in such tasks. Our overall approach to the study of drugs and cognition has been to test the acute effects of the drugs on cognitive processes that can reasonably be assumed to be required for the execution of a broad range of tasks. Once a vulnerable stage is identified, we have attempted to confirm the result using constructive replications and additional task-related experimental variables. Thus, our specific aim was to discover drug effects that could be generalized to a broad range of human performance tasks.

Previous studies in our laboratory (Tharp et al., 1974) and by Huntley (1974) indicated that a major target of alcohol was the information processing stage of "response selection". Probably included in this stage is the operation of "stimulus-response translation" (Teichner & Krebs, 1974). The information processing studies conducted under this contract used reaction time (RT) procedures to test the notion that alcohol impairs the functioning of this limited-capacity translation mechanism. Alcohol appears to impair RT in every low (stimulus-response) compatibility tested: (1) with unfamiliar S-R pairings, (2) with spatially incompatible responses (Robinson & Peebles, 1974) and (3) with high translation load. In general these experiments indicate that alcohol slows RT in proportion to the stimulus-response translation load imposed by the task.

The studies of alcohol and memory were targeted on the possible effects of the drug on organizational processes, retrieval mechanisms, and encoding (depth of processing). The tasks included free recall and tests of recognition. Overall the data suggested that organizational or associational processes in long-term storage were particularly vulnerable to alcohol intoxication. There was no evidence to support the notion that alcohol intoxication impairs memory scanning. Some of the data suggested that retrieval operations may be vulnerable to intoxication, but the evidence was not strong. Likewise there was some evidence that intoxicated subjects apply a more stringent criterion for identifying previously presented items (increased β).

Studies of the effects of acute alcohol on sleep patterns of 47-63 year old normal males were conducted to allow comparison with age-matched alcoholics. Some enhancement of delta sleep was seen in each of the normals, suggesting that the mechanisms for induction of delta sleep may be relatively intact in normal subjects up to advanced middle age. Alcohol failed to potentiate delta sleep in our older chronic alcoholics.

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EFFECTS OF ALCOHOL ON HUMAN PERFORMANCE AND SLEEP

Final Comprehensive Report

Boyd K. Lester, M.D., and Harold L. Williams, Ph.D.

Supported by

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Table of Contents

Effect of Alcohol on Human Performance and Sleep

I. Studies Focused on Information Processing.....3

A. Experiment 1: Reading vs. Shadowing.....4

B. Experiment 2: Reading vs. Picture Naming.....5

II. Studies Focused on Memorial Processes.....11

A. Past Research within the Storage Model.....13

B. Recent Research.....14

C. Results and Discussion.....14

1. Memory of 15-word lists of English nouns.....14

a. Immediate Free Recall.....15

b. Recognition Task.....15

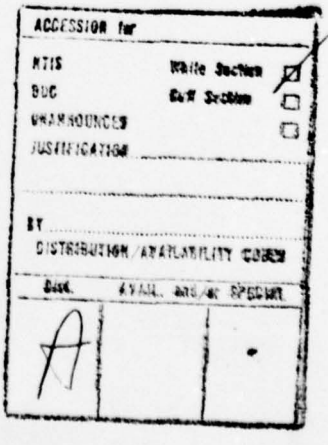
c. Immediate Recognition.....15

d. Final Free Recall.....17

2. Intra-List Recognition.....17

3. Multi-Trial Free Recall of Categorized Lists.....17

III. Studies Focused on Sleep.....21



List of Illustrations

Figure 1. Verbal Reaction Time for Visual and Auditory Words Under Baseline and Alcohol Conditions.....6

Figure 2. Verbal Reaction Time for Visual and Auditory Words as a Function of Log Word Frequency Under Baseline and Alcohol Conditions.....7

Figure 3. Verbal Reaction Time for Printed Words as a Function of Log Word Frequency on Alcohol and Baseline Sessions.....9

Figure 4. Verbal Reaction Time for Pictures as a Function of Log Word Frequency on Alcohol and Baseline Sessions.....10

Figure 5. Serial Position Functions for Immediate Free Recall. Main Effects of Alcohol and Serial Position were Highly Significant.....16

Figure 6. Serial Position Functions for Final Free Recall Summed Across the 10 Lists.....16

Figure 7. Final Free Recall. The Functions Represent the Number of Words Recalled from each of Ten 15-Word Lists.....18

Figure 8. Interaction of Alcohol and List Arrangement with Inter-trial Repetitions as the Dependent Variable.....20

SUMMARY

Effects of Alcohol on Human Performance and Sleep

This contract supported two investigations of the effects of alcohol on humans. The primary project was the investigation of the effects of alcohol on information processing and memory. The second project was the phase-out of our studies of the effects of acute and chronic alcohol ingestion on physiological sleep patterns.

The long-term goal of this research has been to increase our knowledge of the effects of alcohol on human performance. The long-term expectation is that analysis of the effects of alcohol on information processing tasks will reveal the locus of alcohol effects on the different hypothetical cognitive processes involved in such tasks. Our overall approach to the study of drugs and cognition has been to test the acute effects of the drugs on cognitive processes that can reasonably be assumed to be required for the execution of a broad range of tasks. Once a vulnerable stage is identified, we have attempted to confirm the result using constructive replications and additional task-related experimental variables. Thus, our specific aim was to discover drug effects that could be generalized to a broad range of human performance tasks.

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Effects of Alcohol on Human Performance and Sleep

I. Studies Focused on Information Processing (High Accuracy Procedures)

Recently, we reported the results of a series of experiments that examined the effects of alcohol on speed and accuracy in three character-recognition tasks (Rundell, Tharp, Lester and Williams, 1973; Tharp, Rundell, Lester and Williams, 1974). Stimulus discriminability, stimulus-response compatibility (SRC), number of stimulus-response alternatives (N_A), stimulus probability, and categorizing requirements were varied orthogonally with alcohol in an attempt to apply differential load to three hypothesized stages of information processing: stimulus preprocessing (and encoding), stimulus categorization and response selection processes. Examination of patterns of additivity and interaction among these experimental variables revealed consistent positive interactions between the effects of alcohol and those of treatments such as N_A and SRC, intended to influence information outputting operations. The effects of stimulus discriminability, a treatment directed toward stimulus preprocessing and encoding processes were invariably additive with those of alcohol and the other experimental variables. We concluded that in simple character-recognition tasks, alcohol causes specific impairment in information outputting operations, such as those involved in response selection. Huntley (1974), employing choice reaction time (CRT) tasks, and similar experimental variables, arrived at the same general conclusion. Patterns of statistical relationships were somewhat different in the two sets of experiments, but our studies and his agreed that the positive interaction between alcohol and N_A increased with low stimulus-response compatibility. These results indicate that tasks requiring high-speed, relatively incompatible responses to low-probability stimuli are particularly sensitive to alcoholic intoxication.

Our perspectives on this work have changed somewhat after reading the excellent analytical review of visual CRT by Teichner and Krebs (1974). For example, their quantitative analyses of most of the CRT literature, and their own experiments on simple visual RT indicate that N_A has some influence on each of the cognitive stages implicated in CRT. Furthermore, they developed a very convincing argument that the hypothetical component processes in choice reaction time should include a stimulus-response translation operation (T_{S-R}) located between the stimulus categorization and response selection stages. Welford had arrived at a similar conclusion in 1968. Teichner and Krebs' logical analysis of two CRT tasks, Light-Key and Digit-Key, derived the implication that the latter task contains a requirement for translation that is not a component process of the former. The Light-Key task is a spatial matching task in which both the stimuli and the response keys are arranged according to the same left-to-right position code. Once the signal is encoded by position, the response rule is one-to-one. In the Digit-Key task, however, the subject must first encode the numeral, then translate from numerical to position code by a spatial rule, then select the correct motor program and respond. Once the translation process is completed, the response-selection operation appears to be identical for the two tasks. This analysis generated a precise definition of incompatibility between stimulus and response. That is, reduced compatibility exists when the task requires translation from one to another coding dimension. These considerations imply that most SRC treatments exert load on the translation stage rather than on response-selection processes. Therefore, the positive interaction between the effects of alcohol and SRC found both by Huntley

and ourselves may mean that cognitive processes associated with translation rather than with response selection are particularly vulnerable to alcohol.

A number of recent experiments have shown that alcohol has a large effect in verbal RT paradigms which employ visual stimuli (Moskowitz and Roth, 1971; Huntley, 1974; Tharp et al., 1974). The tasks used in these experiments all require a "visual-to-verbal" translation operation. Since translation appears to be particularly vulnerable to the effects of alcohol, we decided to compare several tasks which varied primarily in their "visual-to-verbal" translation requirements, but which held response selection constant.

The stimuli for these experiments were drawn from several word-frequency categories in the Thorndike-Lorge Lists, ranging from high (100 or more) to low (0.9-0.1) word occurrences per million words of written text. For reasons that will become apparent, only nouns with high imagery properties (confirmed by pilot studies) were selected.

A. Experiment I: Reading vs. Shadowing.

The first experiment described here attempted to vary the load on translation operations while holding response selection constant. The two tasks were Word-Shadowing and Word-Reading. In Word-Reading, word stimuli are presented one-at-a-time on a central display, and the subject reads each word aloud as fast as he can. Verbal reaction time is the dependent variable. The subject must see the word, recognize it, translate from a visual to verbal code, select the correct motor-speech program and say the word. In Word-Shadowing the subject must hear the word, recognize it, select the correct motor program and respond. There is no apparent translation requirement.

Since N_A apparently influences all of the component processes of CRT from stimulus encoding to response selection, we suspected that stimulus probability, defined by word frequency (WF) would have a main effect on each task. Furthermore, since Word-Reading contains a translation requirement that is not present in Word-Shadowing, we anticipated a positive interaction between the effects of task and WF. Note, however, that rapid responding to visually presented words is for most young adults, a familiar, highly practiced task. Thus, the differential load on translation operations between the two tasks is probably small.

With response-selection processes equated on these two tasks, we can examine the differential effect of alcohol on translation operations. We anticipated a task by alcohol interaction effect. The combined effects of alcohol and WF were not easily predicted. It will be recalled that the size of the interaction between the effects of alcohol and N_A depends on the level of incompatibility between stimulus and response. It is probable that the Word-Reading task employed here places only a moderate load on translation operations. Thus, we might observe only a trend toward a three-way interaction between task, WF and alcohol.

A group of 24 young males, recruited from nearby colleges and universities, served as subjects (Ss) in this experiment. Each S was tested individually in one of the 24 possible orders for four different stimulus sets. Similarly, each set was presented according to the order randomly assigned to one of four separate sessions: practice, $baseline_1$, alcohol, and $baseline_2$.

Subjects were asked to get a normal night's sleep, and to refrain from taking such stimulants as coffee or coke at least four hours prior to each session. In addition, Ss were required not to eat during the four hours prior to taking alcohol. An alcohol dose of 1.056 g/kg (95% ethanol) was mixed in

a 1:4 ratio with ginger ale. This mixture was divided into three drinks which S consumed in a 30-minute period.

Blood alcohol concentrations (BAC) were measured every ten minutes with a Stephenson Model 900 Breathalyzer. Peak BAC averaged 108 mg%.

The stimuli for both tasks were randomly drawn from the frequency categories described above with the following restrictions: (1) equal numbers of words were drawn from each of the five WF categories and (2) each WF category contained an equal number of one and two syllable words. The auditory words were recorded on audio tape (Scotch #206) and presented to the subject at a rate of one word every 7.5 sec. The onset of each stimulus word triggered a voice key which started a millisecond timer with a triggering accuracy of $\pm$ 3 msec. The onset of the subject's response then triggered the same voice key to turn off the timer. Each visual stimulus was rear-projected on a 3" x 5" screen. Four warning lights surrounding the projected area were illuminated for 1.25 secs prior to each slide presentation. When the warning lights switched off, the slide was projected (Kodak Ektagraphic Projector with Grass Model PS22 Strobe Light Source) and the RT counter started. The S's vocal response triggered the voice key and turned off the RT counter.

Each session consisted of 4 blocks of 25 visual words and 16 blocks of 10 auditory words. Half of the Ss received visual stimuli first on all four sessions while the remaining Ss received auditory stimuli first.

Subjects were instructed to respond as quickly and accurately as possible. To motivate them, one penalty point was administered for each half-second of RT and an additional ten points were added for each erroneous response (including unintelligible sounds). A financial bonus was paid to the S with lowest penalty score on each session.

Figure 1 shows that alcohol increased reaction time in the Word-Reading task by 33 msec, but had almost no effect on the speed of Word-Shadowing. The task by alcohol interaction effect was significant ($p < .05$). Thus, the shadowing task, which is perhaps an analog of the Light-Key task in the visual RT literature, was not sensitive to alcohol.

Figure 2 shows that WF affected both tasks. High-frequency auditory words were shadowed 51 msec faster ($p < .01$) than words from the lowest frequency category. One implication of this finding is that WF, like N_A, probably influences processing components in addition to translation operations.

High-frequency visually presented words were read 84 msec faster than rare visual words, and the task by WF interaction effect was just short of significance at the 0.05 level. There was no trend toward a triple interaction between the effects of alcohol, WF and task ($F < 1$). The additive relation between the effects of alcohol and WF suggests that the two treatments were influencing separate processing stages in the tasks, but the data do not suggest which stages may be differentially involved.

B. Experiment 2: Reading vs. Picture Naming.

Experiment 2 varied the load on component processes involved in categorization and stimulus-response translation, but at a higher level of cognitive difficulty than Experiment 1. Subjects were presented on alternate trial blocks with pictorial and word stimuli again selected from Thorndike-Lorge word lists such that the words varied with respect to their probability of occurrence in English text. The subject's task was to name each picture and read each word as rapidly as possible, while a voice key and associated electronics recorded RT.

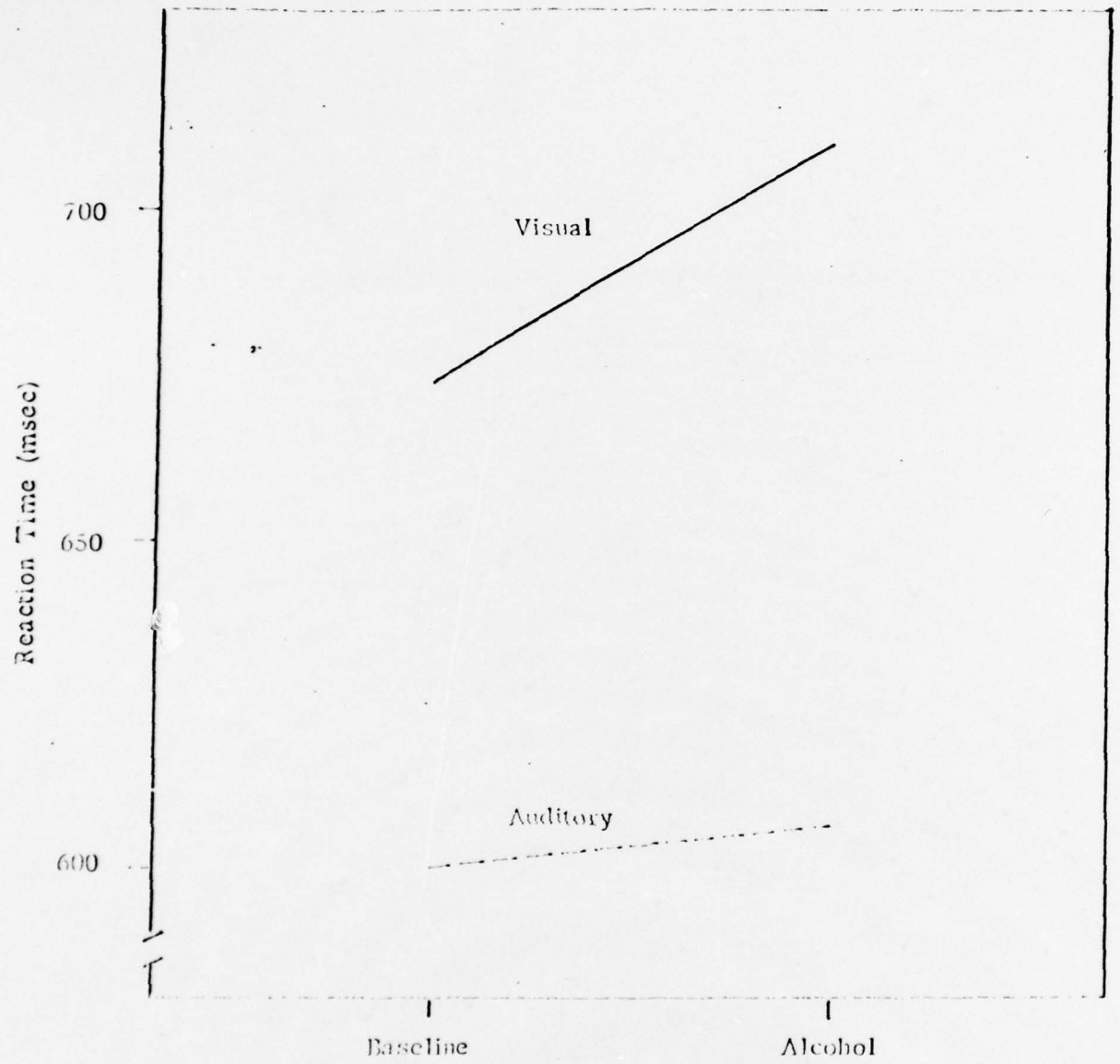

Figure 1. Verbal Reaction Time for Visual and Auditory Words Under Baseline and Alcohol Conditions

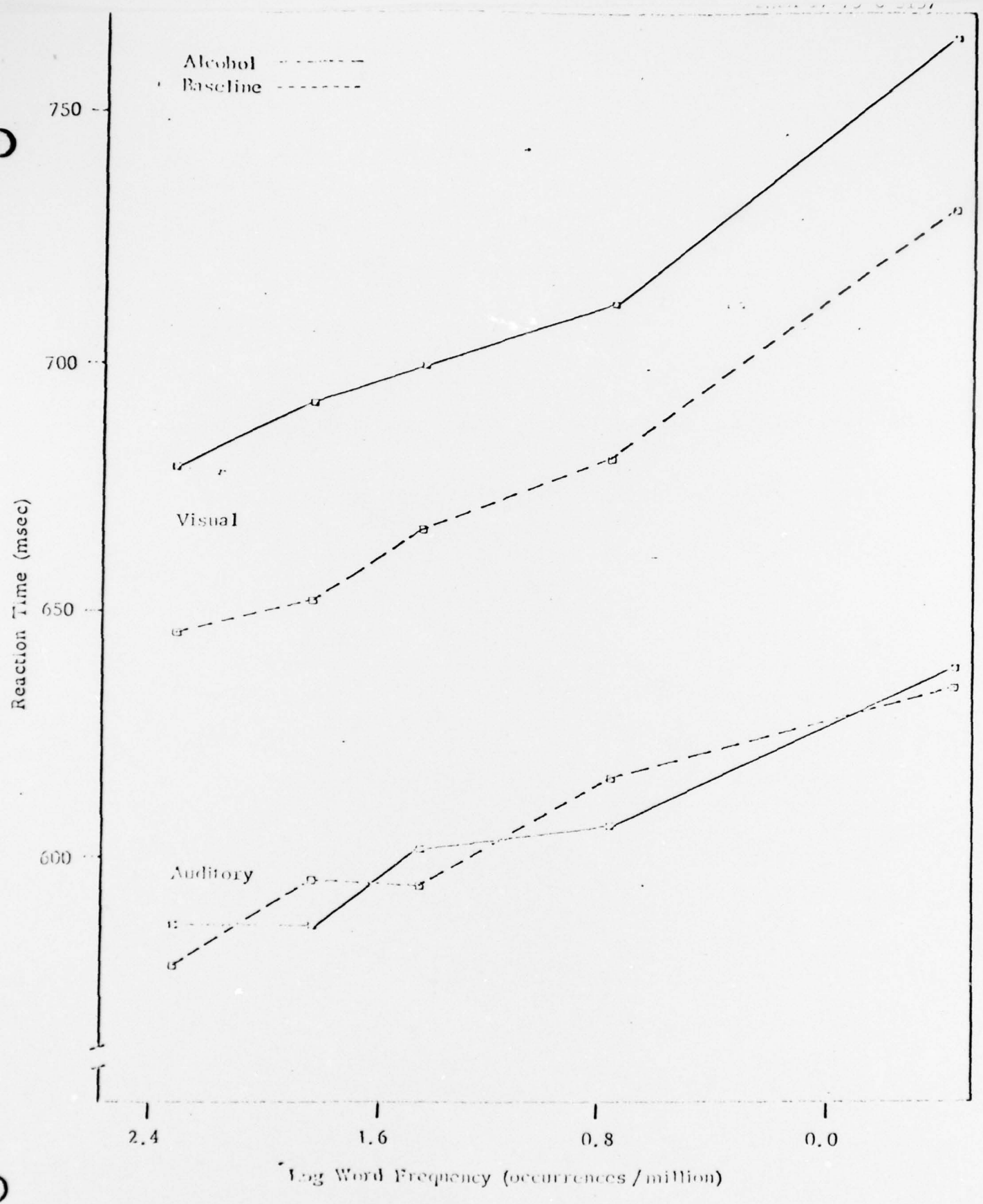

Figure 2. Verbal Reaction Time for Visual and Auditory Words as a Function of Log Word Frequency Under Baseline and Alcohol Conditions

The Word-Reading task was the same in all essentials as that described in the first experiment. It will be recalled that both alcohol and WF caused slowing on that task although their effects did not interact. Picture-Naming must contain a complex series of component processes. The subject must see the picture, encode it visually, categorize it, name it, select the correct verbal motor program, and respond. Note also that the naming operation requires the subject to interrogate his memory, a requirement that is at least minimized in the Word-Reading task.

Studies by others indicate that Picture-Naming is strongly affected by WF (Oldfield and Wingfield, 1965) and by alcohol (Moskowitz and Roth, 1971). Moreover, the alcohol effect was greatest with low-probability stimuli. The results of those studies would be confirmed by a strong WF by alcohol interaction effect. Compared to Word-Reading, Picture-Naming places high load on cognitive operations associated with translation, categorization and retrieval from memory. Thus, we should anticipate a substantial three-way interaction between the effects of task, alcohol and WF.

Twenty-four young males, recruited from nearby colleges and universities, served as subjects in this experiment. Each S was tested individually in one of the 24 possible orders for four different stimulus sets. Each set was presented according to the order randomly assigned in one of four separate sessions: practice, baseline$_1$, alcohol, and baseline$_2$. The first three sessions were on consecutive days at the time of day for each S. Baseline$_2$ followed the alcohol session by 48 hours. Management of subjects and alcohol dosage were the same as in the first experiment.

The stimuli consisted of 440 items, divided equally into five word frequency groupings after Moskowitz and Roth (1971). The word frequency categories from high to low were (1) 100 or more, (2) 99-50, (3) 49-11, (4) 10-1.0, and (5) 0.9-0.1 word occurrences per million words of written text. Only one- or two-syllable nouns with high imagery (i.e., capable of being readily identified from an outline drawing by eight pilot Ss) and having few synonyms were selected. Five practice words and five practice pictures began each session. The experimental stimuli which followed consisted of five blocks of 10 words and five blocks of 10 pictures arranged in a balanced design. Two items from each word frequency grouping were randomly assigned to each block.

Each stimulus (a 35 mm slide) was rear-projected on a 3" x 5" screen. The subject, seated about 30" in front of the screen, saw the projected stimuli without needing to make large scanning movements with his eyes. Four warning lights surrounding the projected area were illuminated for 1.25 sec prior to each slide presentation. When the lights switched off, the slide was projected and the RT counter started. The subject's vocal responses triggered a voice key, and after a constant delay of 14 msec, turned off the RT counter. An intertrial interval of 10 sec separated slides.

Instructions to the S concerning accuracy and speed, as well as the penalty points and financial rewards, were the same as for the first experiment.

Figure 3 illustrates that WF again significantly affected ($p < .001$) the time required to read words and that a logarithmic function adequately describes this relationship. Overall, high frequency words were read 98.9 msec faster than rare words. In addition, alcohol slowed RT by an overall 53 msec ($p < .001$), but this impairment was not a function of WF. Thus, again in a task with moderate translation requirements, alcohol, as predicted, caused overall slowing, but this effect was additive with that of WF.

Figure 4 shows that speed of picture naming is also a logarithmic function of WF. Subjects required an average of 848 msec more time to name rare pictures

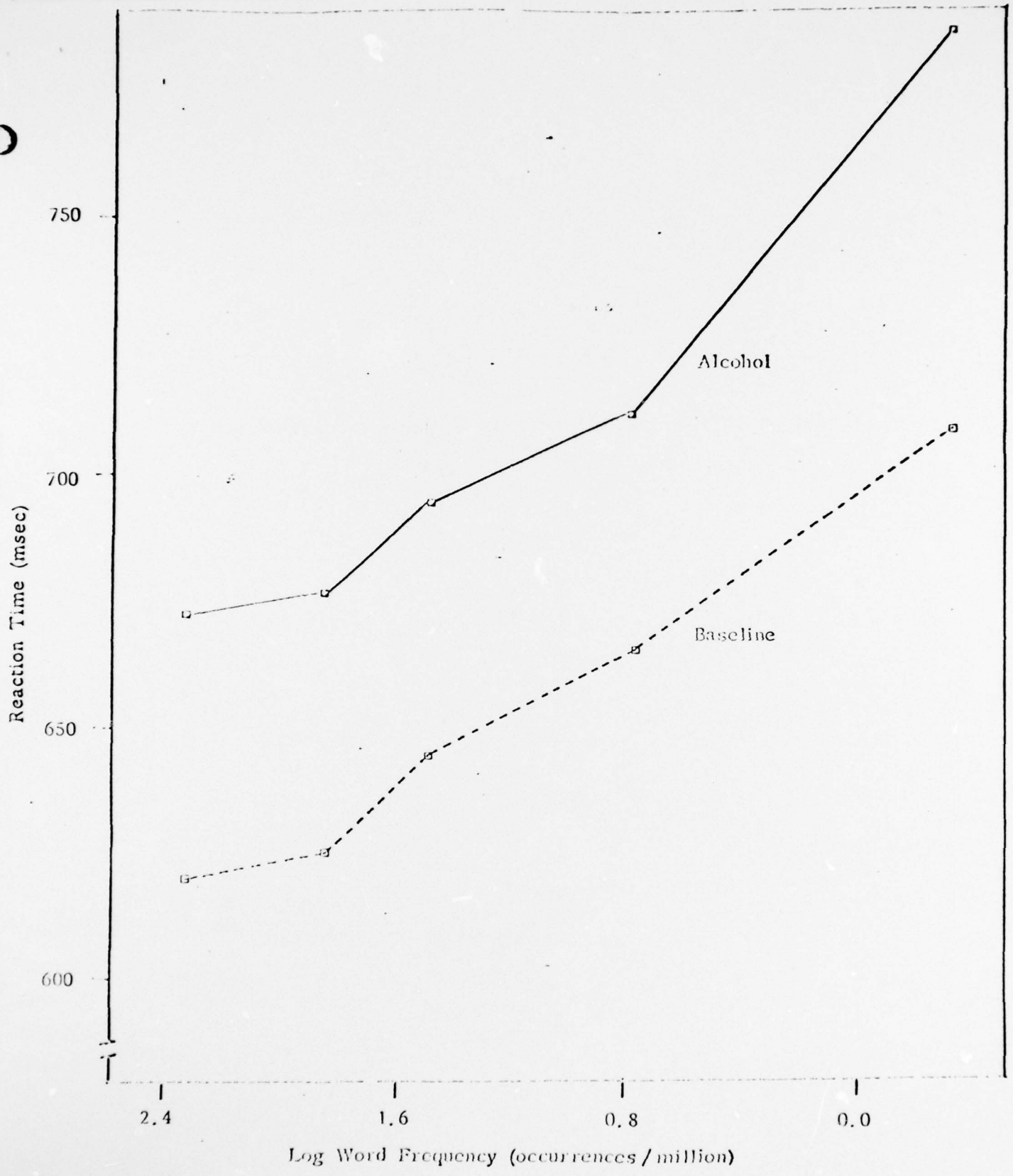

Figure 3. Verbal Reaction Time for Printed Words as a Function of Log Word Frequency on Alcohol and Baseline Sessions

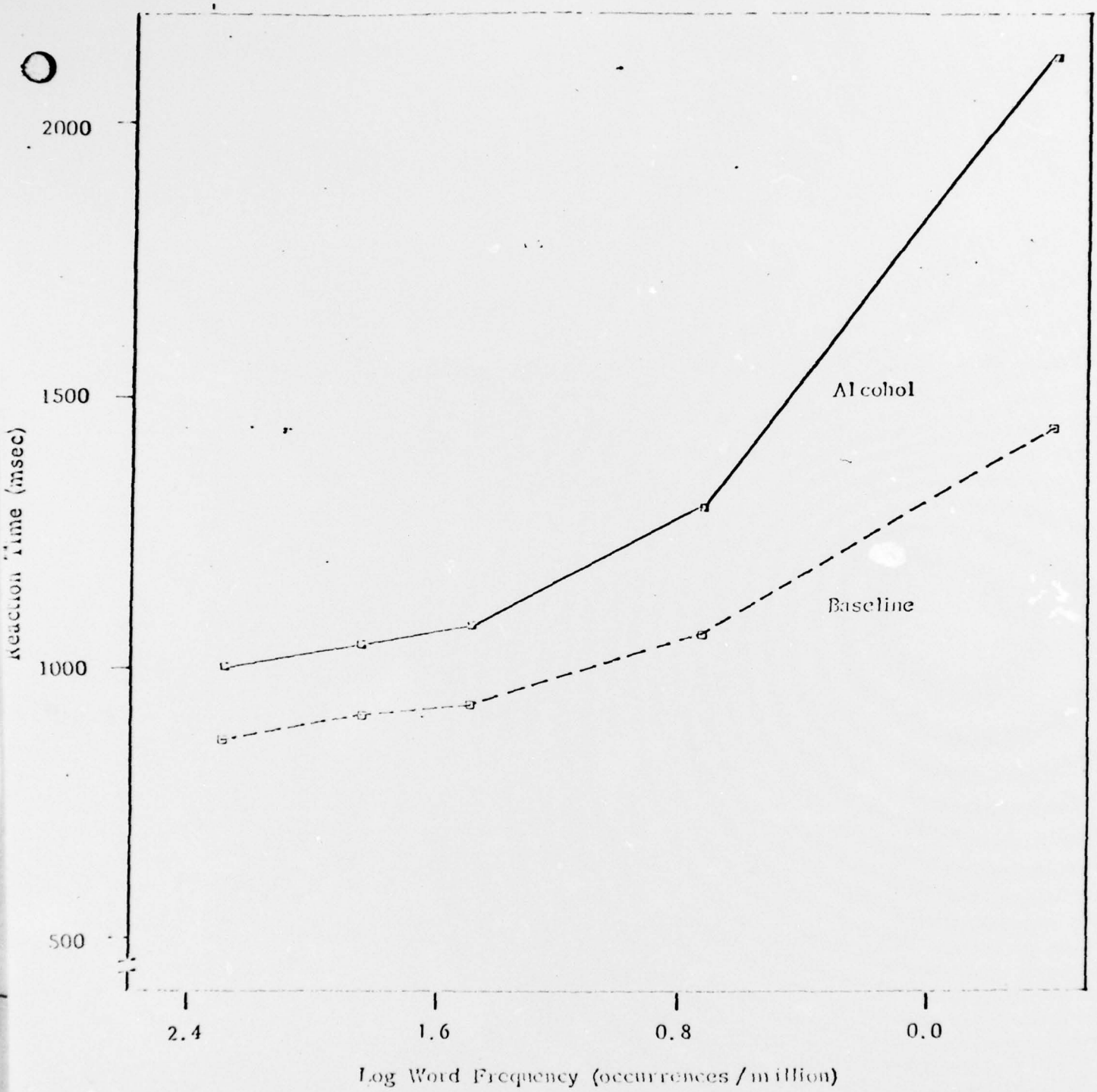

Figure 4. Verbal Reaction Time for Pictures as a Function of Log Word Frequency on Alcohol and Baseline Sessions

than to name common ones ($p < .001$). In addition, alcohol slowed RT over the five word frequency categories by an overall 175 msec ($p < .001$). As anticipated the size of the alcohol effect was a function of WF. Alcohol increased RT to low frequency stimuli by 340 msec more than to common stimuli ($p < .01$).

The overall RT difference for pictures drawn from low- and high-frequency word categories was about 750 msec more than the RT difference for reading low- and high-frequency words ($p < .001$). Finally, the three-way interaction between task, WF and alcohol was significant.

In summary, the results of these studies are consistent with the view that word frequency, like N_A, influences all of the component processes of choice reaction time. Furthermore, as with N_A, the effects of WF increase with increasing load on such central processes as categorization and stimulus-to-response translation. Alcohol had no effect on verbal RT in an auditory shadowing task for which translation operations were minimized. Alcohol did slow performance on the Word-Reading task which does contain a visual to verbal translation requirement. However, the anticipated interaction between the effects of WF and alcohol did not occur. Interaction between these two treatments occurred only at the level of Picture-Naming.

These findings confirm the results of earlier studies by Oldfield and Wingfield (1965), and Moskowitz and Roth (1971). Furthermore, they confirm Huntley's (1974) finding that the size of the interaction between the effects of alcohol and stimulus probability depends on the load placed on other processes such as stimulus-response translation.

Unfortunately, however, the Picture-Naming task is so complex that no convincing argument could be made concerning the probable locus of alcohol effects on information processing.

The data from these studies are consistent with the conclusions of Moskowitz (1973) that alcohol appears to influence a limited-capacity mechanism. The data summarized above, together with the findings of Tharp et al. (1974) and Huntley (1974), specifically imply that alcohol impairs "response selection" or "translation". Alcohol impaired RT in every low-compatibility condition tested: (1) with unfamiliar stimulus-response pairings, (2) with spatially incompatible responses (Robinson & Peebles, 1974), and (3) with high translation load. Since stimulus-response compatibility influences the "translation" or "response selection" process (Sternberg, 1969; Smith, 1961; Teichner & Krebs, 1974), the patterns of interaction between alcohol and this variable suggest that the drug affects this stage.

II. Studies Focused on Memorial Processes

During the past several years our investigations of the effects of alcohol on human performance and cognitive functioning have included a number of experiments focusing on memory. To some extent, these studies have incorporated memorial operations (e.g., memory scanning) within the broader context of information processing. At the same time, however, we have developed an independent line of research with the primary emphasis on memorial processes. For the most part, this group of studies has utilized variations of the free recall paradigm in an attempt to isolate those mechanisms of memory vulnerable to alcohol intoxication.

The current state of experimental work dealing with alcohol and memory is such that little of what is known can be related to general theories of memory. The majority of published studies centering on the effects of alcohol on human memory are empirical or "task-specific" in nature (e.g., Carpenter &

Ross, 1965; Kalin, 1964; Storm & Caird, 1967). Since little work has been carried out within a theoretical framework, the knowledge obtained is often difficult to generalize to other tasks and other experimental situations. Perhaps more importantly, such studies yield little information that is useful for the general understanding of human memory.

In the hope of circumventing some of the shortcomings of earlier atheoretical studies, we began a series of studies within the framework of one of the major classes of models of human memory: "storage models" (after Glanzer, 1972). Storage models of one type or another have been described by the following investigators: Anderson (1972), Atkinson and Shiffrin (1965, 1968, 1971), Bower (1967a,b), Broadbent (1969, 1970, 1971), Glanzer (1972), Kintsch (1970), Laughery (1969), Norman (1968, 1969), Norman and Rumelhart (1970), Shiffrin and Atkinson (1969), Waugh and Norman (1965), and Wickelgren (1970). The primary distinguishing feature of this class of models is the characterization of human memory as consisting of multiple, functionally discrete memory stores. In general, these models arrange memory stores in a sequential organization, and information is transferred from one store to another. The memory stores in storage models are assumed to vary with regard to storage capacity, read-in/read-out rates, mechanisms of address, and the time constants associated with forgetting (Murdock, 1974).

The evidence for multiple storage mechanism comes from a broad series of studies extending over the past decade or so and including both normal and braindamaged subjects (see, for example, Anderson and Bower, 1972; Atkinson and Shiffrin, 1968; Baddeley and Warrington, 1970; Broadbent, 1958, 1968; Cermak and Butters, 1973; Craik, 1968, 1970; Drachman and Arbit, 1966; Glanzer and Cunitz, 1966; Murdock, 1967, Norman, 1968; Sperling, 1963, 1967; Waugh and Norman, 1965). As one might imagine, within the class of storage models there is a good deal of disagreement regarding the precise nature (and number) of the postulated memory stores. Nevertheless, it is possible to specify what might be considered a "modal" storage model (see also, Murdock, 1967). Such a model might include three distinct memory stores: (1) a perceptual or sensory store, (2) a short-term store, STS, (primary memory), and (3) a long-term store, LTS, (secondary memory). Generally input to these stores is considered to be sequential.

The perceptual store (Sperling, 1960) is a very short-term buffer memory which receives incoming sensory information for subsequent read-out into STS. Short-term store can be conceptualized as a limited capacity buffer storage system which may hold information for various types of processing (i.e., an operating register) or for transfer to LTS. Whereas information is lost from the perceptual store by decay (maximum storage time $\simeq$ 250 msec), information is lost from STS by displacement (Raymond, 1969; Shiffrin, 1970; Reitman, 1971). Estimates of the capacity of STS range from two or three items (Glanzer, personal communication; Murdock, 1972), up to five or seven items (Atkinson & Shiffrin, 1968; Anderson & Bower, 1972). Information is transferred to LTS from STS, although the transfer does not imply a loss of information from STS. In recall, information in STS is directly accessible, whereas LTS information must be brought into the STS buffer for emission or subsequent processing. (Whether LTS information is directly accessible from the standpoint of STS is a major source of disagreement.) Storage in LTS is usually considered to be permanent, at least once consolidation is complete. Forgetting may occur as the result of failure to store in LTS or failure of the retrieval process. Failure to store an STS item in LTS could be caused by an impairment of the consolidation process or by some load (e.g., speed) on the STS-to-LTS transfer process.

The Atkinson-Shiffrin version of the storage model postulates an LTS retrieval process consisting of search, retrieval, decision, and response operations for recall from a self-addressing LTS. Thus, an LTS location is interrogated and an item is retrieved (entered in STS). While the item is in STS, a comparison is made between the item and the recall stimulus. A probabilistic process governs the decision to emit the item or continue the search. Forgetting could result from a number of possible sources; although, the most sensitive operations are probably involved in (1) finding the proper locations to interrogate and (2) the decision process. The LTS interrogation process, for example, would be greatly hindered if items were not well organized, encoded, or tagged. In other words, if LTS is primarily a self-addressing store, and if the initial search of likely "locations" is a directed operation, then the better the target item is catalogued, the more efficient the initial search process (i.e., the greater the probability that the initial location interrogated will contain the target item). If the target item is retrieved on the initial memory interrogation, then the chance of beta error is reduced, as is the probability of deciding to terminate a "futile" search. Thus, inadequate encoding, tagging, or organization of to-be-remembered items may lead to reduced correct recall scores, increased errors of commission, and slowed emission of responses.

A. Past Research within the Storage Model.

During the past two years, we have completed three experiments examining the effects of acute alcohol intoxication within the storage model. The first two experiments employed the standard free recall paradigm with lists of English nouns. The third experiment of the series involved a free recall learning task. These experiments were directed towards the isolation of the effect of alcohol (1) within specific memory stores (viz., STS, LTS), and (2) to specific operations associated with the stores (e.g., STS-to-LTS transfer). The several task variables utilized in these experiments produced results consistent with the findings of other investigators (Dallet, 1964; Glanzer, 1969; Glanzer & Cunitz, 1966; Glanzer, Gianutsos & Dubin, 1969; Glanzer & Schwartz, 1971; Murdock, 1962; Phillips, Shiffrin & Atkinson, 1967; Posner & Rossman, 1965; Postman & Phillips, 1965; Puff, 1966; Raymond, 1969; Reitman, 1971; Shiffrin, 1973; etc.). In each experiment the alcohol effect (BAC = 90-100 mg percent) on overall recall was highly reliable ($p < .001$). However, in none of the experiments was it possible to isolate the alcohol impairment to a single store. The expected interactions which would permit the acceptance of a localized effect were consistently small ($F_{2,60} = 0.5$; $F_{2,32} = 0.7$; and $F_{2,60} = 0.2$). Moreover, estimates of the effect of alcohol intoxication on recall from STS were made using three different methods of calculating STS. In each case there was some evidence for rejecting the null hypothesis ($t_{30} = 1.85$, $p < .10$; $F_{1,17} = 4.26$, $p < .10$; $t_{18} = 3.25$, $p < .01$; $t_{36} = 1.93$, $p < .10$).

For the first two experiments, involving the (single trial) free recall paradigm, task variables were employed to load the STS-to-LTS transfer operation and to clear list items from STS. Neither variable responded differentially to the two levels of the alcohol variable. A third task variable, list length (12 or 18 words), did interact with the alcohol variable such that the intoxicated subjects were particularly impaired for the longer lists. Of the several possible interpretations of the alcohol x list length interaction, one of the most attractive was that alcohol had impaired the degree of organization of

items in LTS. An organizational impairment might be expected to interfere with retrieval by making the location of the proper memory addresses more difficult (see above). Under such conditions one would expect decreased recall (confirmed), increased errors of commission (partially confirmed), and increased latency of response (untested).

The third experiment of the series attacked one form of the organizational hypothesis by varying the strength of associational linkages in a standard free recall learning paradigm and measuring the amount recalled, subjective organization (Tulving, 1962), stability of recall from trial to trial, and sequential organization of recall (cf., Bousfield & Bousfield, 1966). Briefly, the results of this experiment indicated that intoxicated subjects were impaired in the use of associative linkages, especially when the associative values were low. In addition, there was evidence from the stability of recall measure that the accessibility of items stored in LTS was impaired by alcohol and that the impairment was independent of the associative strength variable. This finding suggested that the intoxicated subjects may have been experiencing retrieval difficulties in addition to the effects of LTS organizational or associative disruptions.

B. Recent Research.

In brief, three previous studies in our laboratory had indicated a strong effect of alcohol intoxication on recall of words from long-term storage. Data from these experiments also suggested an alcohol effect on recall from short-term storage; although, this impairment may be somewhat less substantial than the LTS effect. Some tentative alternative hypotheses, derived from the initial studies were as follows:

1. Alcohol impairs recall by interfering with the retrieval process (e.g., the scanning operation may be impaired).
2. The intoxicated individual tends to encode items at levels less effective for retrieval (i.e., at acoustic rather than semantic levels).
3. Alcohol impairs the individual's ability to effectively organize items in memory. The organizational deficit may occur during storage, or during retrieval, or both.

To test some of these notions, a fourth study in the series was subsequently proposed to investigate the effects of a moderate dose of alcohol (BAC $\simeq$ 100 mg percent) (1) under varying levels of retrieval load, (2) on levels of encoding, and (3) on organization of to-be-remembered items in memory. Upon completion of the experiment (44 subjects), the data was analyzed with the results reported below.

C. Results and Discussion.

1. Memory of 15-word lists of English nouns. Following presentation of each list, Ss performed written free recall (immediate free recall) followed by a recognition task. Since recognition tasks are generally presumed to eliminate memory scanning operations, the comparison of alcohol effects on recall and recognition tasks permit inferences regarding the effect of alcohol on the scanning aspect of retrieval. After all 10 lists had been presented, Ss performed a final free recall task in which they attempted to recall all 150 words.

a. Immediate Free Recall. Alcohol produced a reliable ($p < .001$) decrement in recall (approximately 18%). The serial position curves for both alcohol and placebo conditions were of the typical U shape (see Figure 5); and the F-ratio for serial position was highly significant ($p < .001$). However, the interaction between alcohol condition and serial position was not significant ($F_{4,76} = 1.13$), and the simple main effect of drug was significant for the final three list positions ($p < .05$). These results confirmed previous studies in our laboratory, and they imply that alcohol impairs the recall of items in both long- and short-term storage.

b. Recognition Task. The 15 original list items were presented along with 30 distractors. The subject circled a number from 1 to 6 to indicate his degree of confidence that each item was or was not a member of the previously presented list. For both sober and intoxicated subjects, nearly twice as many items were correctly identified in the recognition task as were recalled in the immediate free recall task. However, alcohol reliably ($p < .001$) reduced the number of correct recognitions. The intoxicated subject tended to commit fewer false recognitions (false alarms), although this result was not quite significant at the .05 level. There was no significant difference between the amount of alcohol-related decrement in the immediate free recall task and the recognition task. This was true whether decrement was calculated on the basis of number of words ($t = 1.03$) or on proportional change ($t = .48$). Thus, there was no indication that the retrieval operation of scanning was the target of the alcohol effect. The signal detection statistics, d' and β were calculated for both alcohol and placebo conditions of the recognition task. The d' estimates were lower in the alcohol condition, again indicating a reduced ability to identify list items ($p < .021$, sign test). A significantly high β statistic for the alcohol condition ($p < .006$, sign test) indicated that when intoxicated, our subjects were more "cautious", i.e., less willing to identify recognition items as being list items. Conversely, once committed to a response, our subjects tended to have a higher degree of confidence in their responses when intoxicated than when sober ($t_{19} = 2.07$, $p < .10$). Indeed, the degree of confidence was significantly higher in the alcohol condition ($p < .05$) for each of three response categories: *hits, correct rejections and misses.* Only for false alarms were intoxicated subjects less confident than sober subjects. However, this latter trend was not statistically significant ($t_{19} = 1.22$).

c. Immediate Recognition. The possibility remained that the delayed recognition task failed to show a disproportionate improvement over immediate free recall performance in the alcohol condition because recognition followed recall. Therefore, twenty-three additional subjects performed the recognition task immediately after list presentation without intervening free recall. The impairment in immediate correct recognitions by the alcohol group (-15%) was similar to that found with recognition following free recall. However, with independent groups and smaller sample sizes, the alcohol effect did not reach statistical significance with the parametric test ($t = 1.67$). When the number of correct recognitions in the immediate recognition task was compared with those in the delayed recognition task, there was no significant difference for alcohol ($t = 1.04$) or placebo ($t = .95$) conditions. Thus, the rejection of the first hypothesis (i.e., that alcohol interferes with the memory scanning aspect of retrieval) was strengthened.

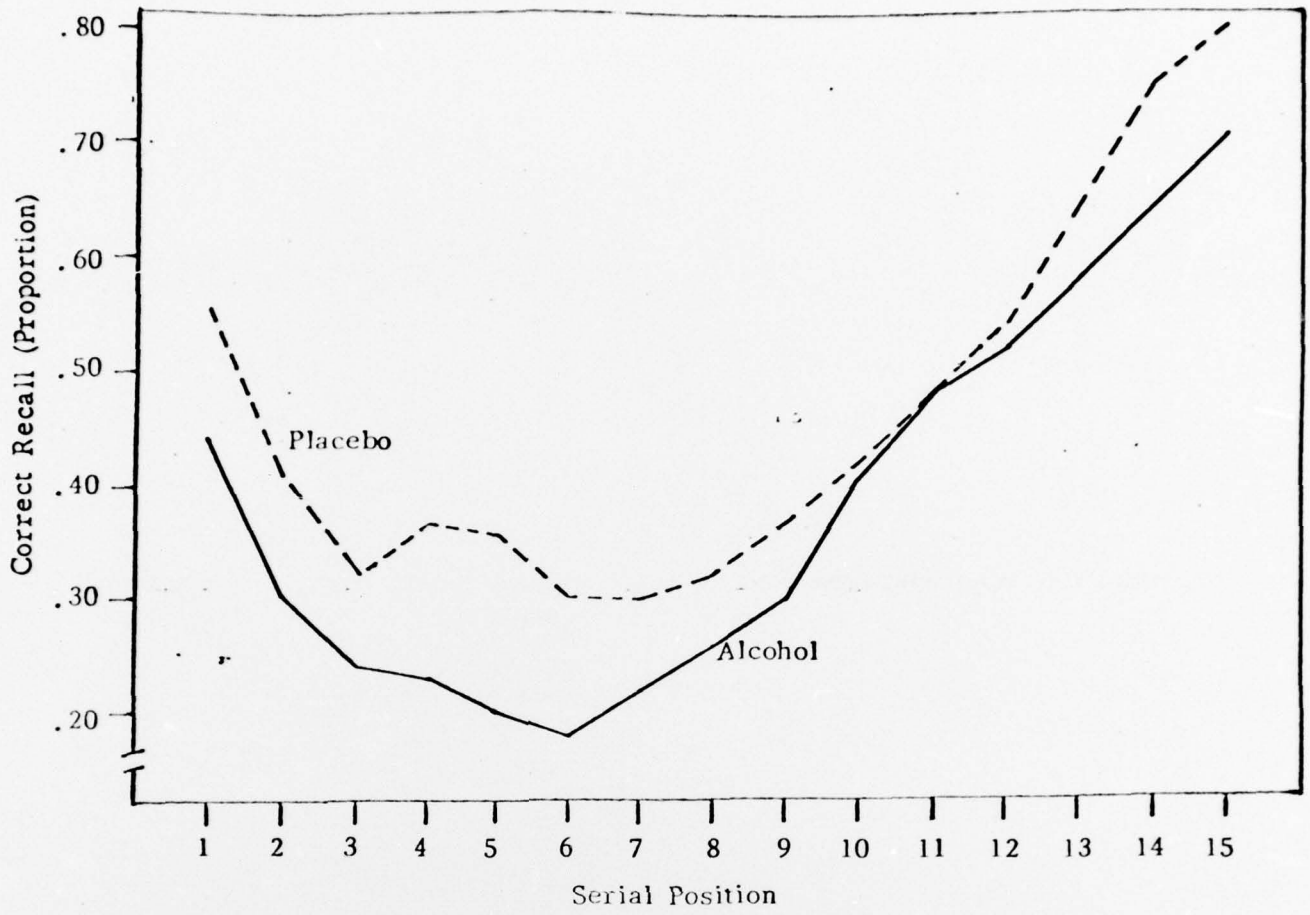

Figure 5. Serial position functions for immediate free recall. Main effects of alcohol and serial position were highly significant.

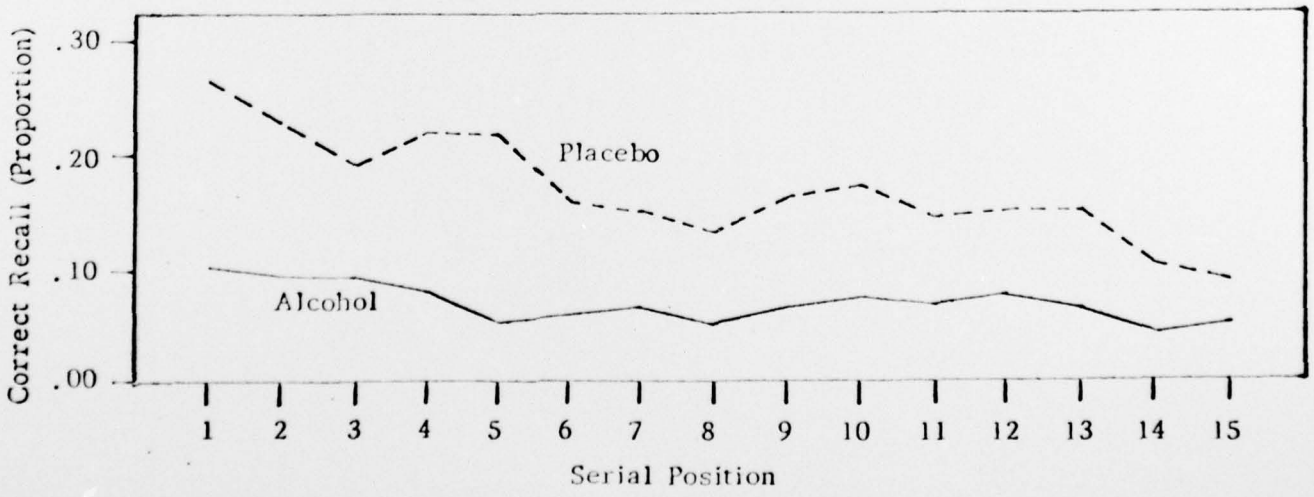

Figure 6. Serial position functions for final free recall summed across the 10 lists.

d. Final Free Recall. Alcohol reduced the number of words correctly recalled ($p < .001$) and increased the number of errors of commission ($p < .05$).

Serial position curves for alcohol and placebo conditions were constructed by combining across lists (see Figure 6). Both curves display the typical negative recency effect. As shown in the figure, alcohol significantly lowered recall at final free recall ($p < .001$), and produced greater recall impairment for final free recall than for immediate free recall ($p < .001$). This finding suggests a possible effect of alcohol on consolidation.

A second set of curves was constructed by calculating the mean recall for list and arranging the lists in order of presentation. These data are displayed in Figure 7. Main effects were significant for drug ($F_{1,19} = 54.3$, $p < .001$) and serial position ($F_{9,171} = 13.3$, $p < .001$), as was the drug by serial position interaction ($F_{9,171} = 3.91$, $p < .001$). Differences between the curves were significant at the .01 level or better (F tests) for the following serial positions: 2, 4, 7, 8, 9, and 10. Examination of Figure 7 reveals that the placebo condition's advantage over alcohol steadily increases from positions 6 to 10. An hypothesis that alcohol interferes with consolidation processes would predict that alcohol-placebo differences would be greatest for the initial list and least for the final list. The present data are contrary to such an interpretation. However, we must point out that the interval separating presentation of the initial list and final free recall was only about 45 minutes, and the number of words recalled by the intoxicated subjects was small ($\bar{X} = 10.3$ words). These data, therefore, do not represent a stringent test of a consolidation interference hypothesis.

2. Intra-List Recognition. A list of 80 words was presented at a 3-second rate to two groups of 22 alcohol and placebo subjects. As each word was presented, the subject responded "yes" or "no" according to whether the word had previously appeared in the list. Ten of the 80 words were repeated once. The list also contained 10 pairs each of synonyms, high frequency associates and homonyms. This task was designed to test the second hypothesis: that intoxicated Ss, like patients with Korsakoff syndrome, tend to encode material at less efficient levels (Cermak & Butters, 1973). If the intoxicated subject were to encode at less effective levels, one would expect a decrease in the hit rate. In addition, encoding at the acoustic level rather than the semantic level would produce a confusion of homonyms. Thus, the subject would tend to make more false positive responses to the second member of a pair of same-sounding words (e.g., "bear" and "bare"). Likewise, encoding on an associative dimension would lead to more false positive responses to high frequency associates. Therefore, if the encoding hypothesis were correct, one would expect, as a minimum, fewer hits and more false alarms in the alcohol condition. The alcohol group did have fewer correct identifications ($p < .05$) but only marginally more false alarms than did placebo subjects. However, since the false alarm rate was very low for both groups (placebo = 9.3%, alcohol = 11.3%), this task may not have provided a sensitive test. Among the three types of distractor items, alcohol produced the greatest increase in false positives for the high frequency associates.

3. Multi-Trial Free Recall of Categorized Lists. Subjects were presented with a single 64-word list composed of 4 category titles (e.g., fish, vehicles, etc.) and 15 exemplars from each category. The list was presented twice, each

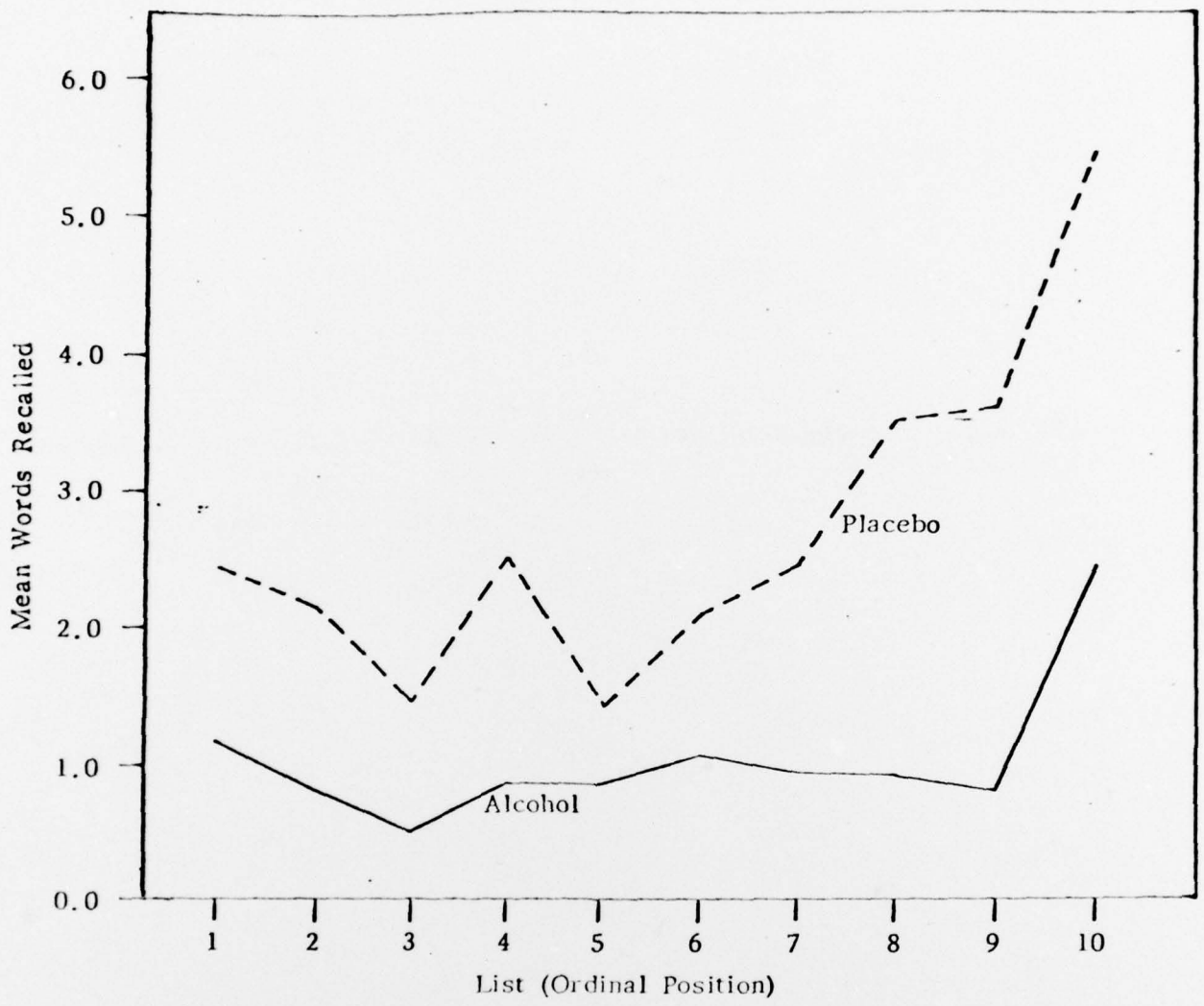

Figure 7. Final free recall. The functions represent the number of words recalled from each of ten 15-word lists.

presentation being followed by free recall. The words were arranged in random order for half the subjects and grouped by category for the remaining subjects. For total recall, significant main effects were found for drug ($p < .001$), trials ($p < .001$), and list arrangement ($p < .05$). The third hypothesis, that intoxicated subjects have difficulty organizing material in memory, leads to the prediction of a positive interaction between the drug and the grouping variable. Although the means were in the predicted direction, the interaction of drug and list arrangement did not reach statistical significance. The trend was for the blocked arrangement to benefit the sober Ss more than the intoxicated Ss. Inter-trial repetitions were calculated to measure sequential constancies in recall from trial one to trial two. This measure is an indicator of subjective organization in memory (Bousfield and Bousfield, 1966). Alcohol decreased sequential organization ($p < .01$), while the blocked arrangement led to increased organization ($p < .01$). Although the rather striking interaction between drug and list arrangement (illustrated in Figure 8) did not reach significance at the .05 level ($p < .10$), a posteriori tests revealed a significant effect of list arrangement for the placebo Ss only ($p < .01$, Tukey's HSD). Thus the data support the notion that both alcohol and the objective organization of the list affect the degree to which items are organized in memory. The Pearson correlation coefficient between inter-trial repetitions and total correct (immediate) recall was .73 ($p < .001$).

Stability of recall was calculated as the proportion of items recalled on Trial 1 that were also recalled on Trial 2 and was analyzed to assess Ss' difficulty in locating the appropriate storage locations in LTS. Alcohol reliably reduced the stability of recall measure ($p < .001$). Although the effect of the list arrangement variable was nonsignificant, there was a significant interaction between drug and list arrangement ($p < .01$). Simple effects analysis revealed a significant list arrangement effect on placebo Ss ($p < .05$) but only a marginal effect for alcohol Ss ($p < .10$). There was also a significant overall correlation of stability of recall with total recall ($r = .49$, $p < .01$).

Z-scores representing category clustering were calculated according to the method recommended by Frankel & Cole (1971). The main effects of drug, trials, and list arrangement were significant at the .001 level in the predicted directions. Although no interactions were significant, a posteriori tests revealed increased clustering in the blocked presentation condition for the placebo Ss (HSD < .01), but the increase was not reliable for the alcohol Ss (HSD > .05). Thus, there was a strong trend for blocking to increase clustering in the placebo Ss, but the results for the intoxicated Ss were more variable. Overall, clustering scores correlated well with total correct recall ($r = .78$, $p < .001$).

In summary, the data do not support the hypothesis that the memory scanning aspect of the retrieval process is affected by alcohol. Stage analysis of the present data, along with previous findings support the notion that alcohol impairs recall from both STS and LTS. The data were not conclusive with the encoding impairment hypothesis. There is a hint that alcohol may affect decision processes such that the intoxicated Ss were applying a more stringent criterion (higher β statistic). The data suggest that, at least in part, the recall deficit may result from an inability to carry out a coherent organizational plan, rather than an inability to formulate a plan. In general, the organizational data are similar to our previous findings. The correlations between recall and the organizational measures were reasonably high, and alcohol seems to undermine this association. Overall our studies have most strongly implicated organization or structuring of memory as a primary target for alcohol.

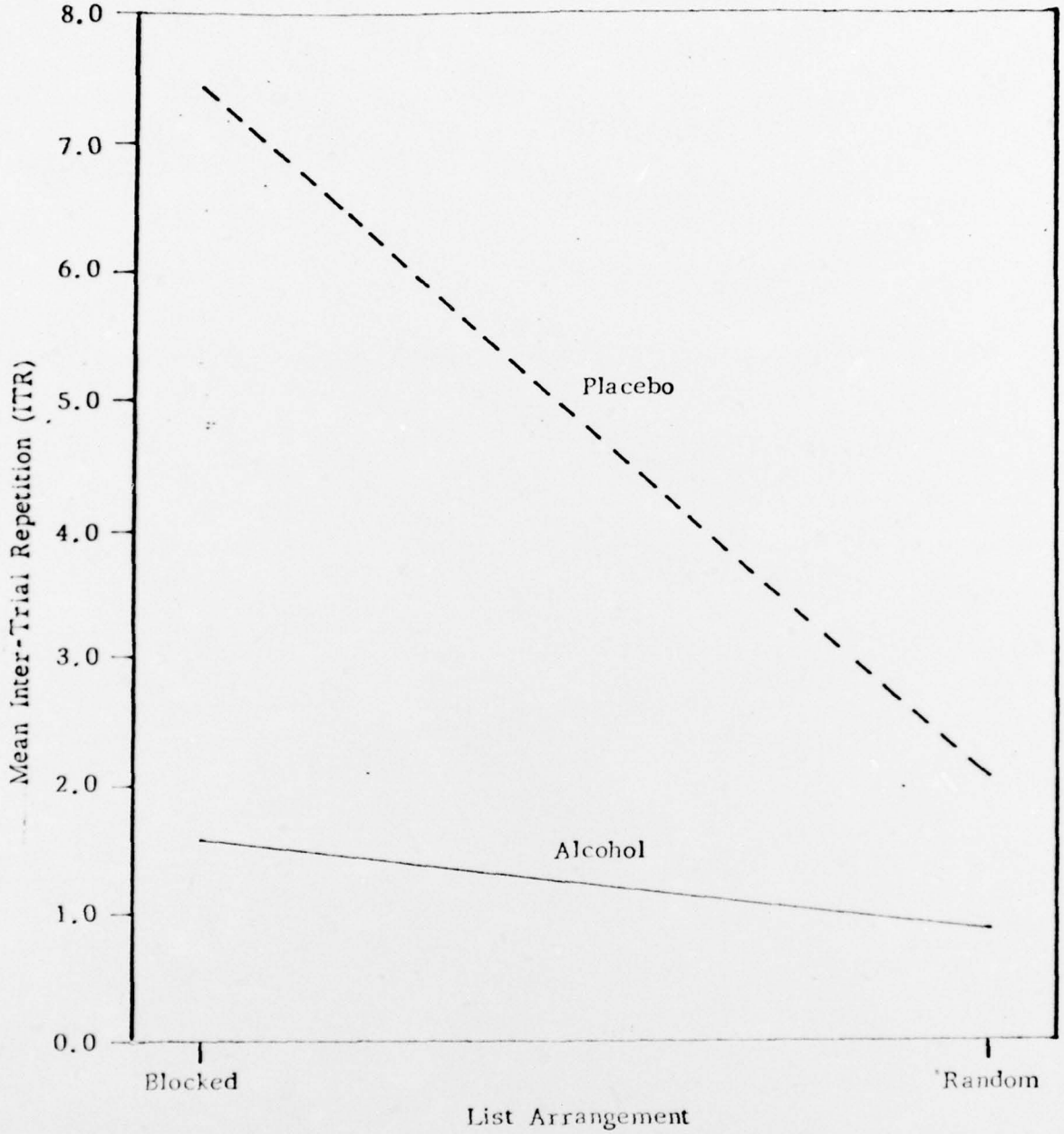

Figure 8. Interaction of alcohol and list arrangement with Inter-trial repetitions as the dependent variable. Lists consisted of four category names and 15 exemplars of each category. Lists were presented by category for the "Blocked" arrangement. The simple main effect of alcohol was not significant for the random arrangement, nor was the simple main effect of list arrangement for the alcohol group.

The sequence of studies summarized above is part of a longer series of experimental probes into the nature of memorial deficit caused by alcohol. There is no question that alcohol causes impaired verbal recall. There has been a strong main effect of alcohol in each of some ten experiments. The question is "Why?" There are strong hints both in earlier experiments and in the series summarized here that the deficit in long-term recall is due to impairment of organizational-associational systems in memory. For example, the intoxicated subject does not spontaneously cluster items by associational linkages as effectively as the sober subject. Moreover, as reported here, the intoxicated subject does not make adequate use of objective list structure for organizing items in memory.

Some of the data can also be interpreted as suggesting an impairment of some aspect of the retrieval process. If the retrieval process, indeed, consists of scanning, item retrieval, and decision operations, our latest study implies that the scanning operation is intact in intoxicated subjects. This suggestion is consistent with data from one of our previous experiments using the Sternberg memory-search paradigm. In that study, too, there was no evidence that the search process was affected by alcohol. If retrieval processes are vulnerable to alcohol, then one might suppose that the decision operation would be the most likely point for future examination. A decision operation such as hypothesized for free recall tasks could be influenced indirectly by several related factors. Among these are (1) the quality of the item retrieved (e.g., the level of encoding) and (2) the organizational and associational context or matrix within which the item is stored. Alcohol could directly influence the decision process (1) by some sort of general impairment or (2) by causing a shift in the criterion for emitting a response or for deciding to terminate the search. There was a hint in the recognition task data (reported above) that subjects set more stringent criteria for recognizing a list item when intoxicated than when sober. Perhaps with a more stringent criterion, it is not surprising that the intoxicated Ss were generally more confident of the accuracy of their responses. Indeed, it is possible that increasing the criterion for emitting a response might lead to a reduction in the number of words recalled.

III. Studies Focused on Sleep

Chronic alcoholism is associated with marked disturbance in the physiologic sleep profile (Johnson et al., 1972; Lester et al., 1973; Lester et al., in press). These disturbances include frequent awakening, particularly from stage REM, acceleration of the ultradian REM cycle, almost total absence of stage 4 sleep, and changes in autonomic levels, represented by increased rates of respiration and of non-specific electrodermal activity. Although these alterations persist for at least several weeks in sober alcoholics, there is very little information on the effects on disturbed sleep of prolonged abstinence. We do not know whether, or to what extent disturbed sleep profiles eventually recover, nor do we know the degree to which age or duration of heavy drinking influence such recovery.

The amount of stage 4 (delta) sleep is diminished in both young (under 40) and older alcoholics, in the severe depressive disorders, and with normal aging. However, it is not certain whether its absence is due to loss of EEG amplitude or loss of the delta (0.5 to 4 Hz) range of EEG frequencies. We recently reported some data showing that loss of EEG amplitude rather than of delta frequencies accounted for the absence of stage 4 in a few chronic alcoholics, but this finding should be checked in a larger sample (Lester et al., in press). The issue has some theoretical importance since the neural processes that control EEG amplitude are probably different structurally and functionally from those that control EEG rhythms.

Moderate doses of alcohol administered prior to sleep tend to potentiate delta sleep in young normal subjects (Rundell et al., 1972), and in young alcoholics as well. However, in older alcoholics, alcohol had no effect on delta sleep (Lester et al., in press). Since alcohol can instate stage 4 in the young alcoholic, one surmises that in these patients the physiological mechanisms for delta sleep are still intact. However, we do not know whether the failure of alcohol to stimulate stage 4 in older alcoholics is really specific to chronic alcoholism or is associated simply with normal aging. This year we undertook studies to examine these issues.

Six normal volunteers with histories of very moderate social drinking (ages 47-63) were studied for four consecutive nights in the sleep laboratory. After one adaptation and two baseline nights, Ss were given drinks consisting of 95% ethyl alcohol mixed with ginger ale. With doses of 1.06 g/kg, BACs ranged from 90 to 110 mg%. Three of the Ss had some difficulty tolerating this dose. Physiological measures included continuous EEG recordings from symmetrical C_4/A_1 and C_3/A_2 placements, submental electromyogram from bipolar placements under the chin, left and right electrooculograms, GSR from palmar surface of middle finger right-hand, heart rate with a lead II EKG placement and respiration from a strain gauge around the solar plexus. Data analysis thus far completed on four of these subjects suggest that on the average they have very little delta sleep, no more than age-matched chronic alcoholics. Each of them, however, showed a small increase in delta sleep during the alcohol night. These trends indicate that the mechanisms for induction of delta sleep may be relatively intact in normal subjects up to advanced middle age. Thus, the failure of alcohol to potentiate delta sleep in the older chronic alcoholic may be related specifically to alcoholism.

A second experiment was undertaken to determine whether physiologic sleep profiles remain abnormal in former alcoholics, totally abstinent for at least two years. Eight volunteers, all successful members of A.A. have been run. Three of these subjects are under 40 and the remaining five are all over 50 years old. Analyses, thus far, suggest that these subjects have EEG profiles compatible with those of normal subjects matched on age. These preliminary results, if sustained, confirm the findings of Adamson and Burdick (1973).

Publications:

Lester, B. K., Rundell, O. H., Cowden, L. C. & Williams, H. L. Chronic alcoholism, alcohol and sleep. In: M. Gross (Ed.), Alcohol Intoxication and Withdrawal: Experimental Studies: Advances in Experimental Medicine and Biology. Vol. 35. New York: Plenum Press, 1973, pp. 261-279.

Lester, B. K., Rundell, O. H., Cowden, L. C. & Williams, H. L. Alcohol and sleep in the chronic alcoholic. N. R. Burch (Ed.), Proceedings of the Seventh Annual TRIMS Symposium. Houston, Texas, 1973. (In press)

Rundell, O. H., Tharp, V., Lester, B. K. & Williams, H. L. Some effects of acute intoxication on information processing stages. Alcohol Technical Reports, 1: 25-32, 1973.

Rundell, O. H. The control of afferency: The central nervous system as a regulator. Biological Psychology Bulletin, 1972, 1, 19-30.

Rundell, O. H., Lester, B. K., Griffith, W. J., and Williams, H. L. Alcohol and sleep in young adults. Psychopharmacologia, 1972, 26, 201-218.

Tharp, V. K., Rundell, O. H., Lester, B. K., and Williams, H. L. Alcohol and information processing. Psychopharmacologia, 40: 33-52, 1974.

Tharp, V. K., Rundell, O. H., Lester, B. K. and Williams, H. L. Alcohol and secobarbital: Effects on information processing. In: M. Gross (Ed.), Alcohol Intoxication and Withdrawal: Experimental Studies: Advances in Experimental Medicine and Biology. New York: Plenum Press, in press.

Williams, H. L., Beaver, W. S., Spence, M. T., and Rundell, O. H. Digital and kinesthetic memory with interpolated information processing. Journal of Experimental Psychology, 1969, 80, 530-536.

Williams, H. L., Tharp, V. K., Rundell, O. H., and Lester, B. K. Some effects of alcohol on information processing. New York: Plenum Press, in press.

Tharp, V. K. Alcohol and Driving Performance: An Information Processing Analysis. Alcohol Technical Reports, Vol. 4, 2, 1975.

Presentations:

Williams, H. L., Rundell, O. H., Spence, M. T., and Lester B. K. Sleep loss: Some physiological correlates of impaired performance. Paper presented at Southwestern Psychological Association meeting, 1968.

Salamy, A., Rundell, O. H., Williams, H. L., and Lester, B. K. The effects of alcohol on the cortical evoked response. Paper presented at Southwestern Psychological Association meeting, Austin, Texas, 1969.

Spence, M. T., Williams, H. L., and Rundell, O. H. The effects of sleep loss and interpolated information processing on short-term verbal retention. Paper presented at Southwestern Psychological Association meeting, Austin, Texas, 1969.

Lester, B. K., Rundell, O. H., Cowden, L. C., and Williams, H. L. Chronic alcoholism, alcohol and sleep. Paper presented at 30th International Congress on Alcoholism and Drug Dependence, Amsterdam, 1972.

Williams, H. L., Tharp, V., Rundell, O. H., and Lester, B. K. Some effects of alcohol on information processing. Paper presented at the NATO Conference on Drugs, Sleep and Performance, Edinburgh, Scotland: 1972.

Lester, B. K., Rundell, O. H., Cowden, L. C., and Williams, H. L. Alcohol and sleep in the chronic alcoholic. Paper presented at the 7th Annual Symposium of the Texas Research Institute of Mental Sciences: Behavior and Brain Electrical Activity, Houston, Texas, December, 1973.

Tharp, V., and Rundell, O. H. Alcohol and secobarbital: Altered states of consciousness assessed by an information processing approach. In, B. M. Jones (Chair), Altered States of Consciousness and Alcohol. Symposium presented at the meeting of the Southwestern Psychological Association, El Paso, Texas, May 1974.

Rundell, O. H., Lester, B. K., and Jones, B. M. Biological rhythms and alcohol effects: Altered rhythms of consciousness. In, B. M. Jones (Chair), Altered States of Consciousness and Alcohol. Symposium presented at the meeting of the Southwestern Psychological Association, El Paso, Texas, 1974.

Tharp, V. K., Rundell, O. H., Lester, B. K., and Williams, H. L. Alcohol and secobarbital: Effects on information processing. Paper presented at the International Congress on Alcoholism and Addiction, Manchester, England, June 1974.

Williams, H. L., Rundell, O. H., and Tharp, V. K. Drinking and information processing in man. Paper presented at the Oklahoma Symposia on Drinking Behavior and Alcoholism, Oklahoma City, Oklahoma, November 1974.

Papers in preparation:

Rundell, O. H., and Williams, H. L. Alcohol and verbal free recall.

Jones, B. M., Rundell, O. H., and Parsons, O. A. Psychophysiological correlates of alcoholism. In R. E. Tarter and A. A. Sugerman (Eds.) Alcoholism: Interdisciplinary approaches to an enduring problem. Reading, Massachusetts: Addison-Wesley.

References

Adamson, J. & Burdick, J.H. Sleep of dry alcoholics. Archives of General Psychiatry, 28: 146, 1973.

Anderson, J.R. FRAN, a simulation model of free recall. In: G.H. Bower (Ed.), The Psychology of Learning and Motivation: Advances in Research and Theory. Vol. 5. New York: Academic Press, 1972, pp. 315-378.

Anderson, J.R. & Bower, G.H. Recognition and retrieval processes in free recall. Psychological Review, 79: 97-123, 1972.

Atkinson, R.C. & Shiffrin, R.M. Mathematical models for memory and learning. Technical Report No. 79, Institute for Mathematical Studies in the Social Sciences, Stanford University, 1965.

Atkinson, R.C. & Shiffrin, R.M. Human memory: A proposed system and its control processes. In: K. W. Spence and J. T. Spence (Eds.), The Psychology of Learning and Motivation: Advances in Research and Theory. Vol. 2. New York: Academic Press, 1968, pp. 89-195.

Atkinson, R.C. & Shiffrin, R.M. The control of short-term memory. Scientific American, August, 1971, pp. 82-90.

Baddeley, A.D. & Warrington, E.K. Amnesia and the distinction between long- and short-term memory. Journal of Verbal Learning and Verbal Behavior, 9: 176-189, 1970.

Bousfield, A.K. & Bousfield, W.A. Measurement of clustering and of sequential constancies in repeated free recall. Psychological Reports, 19: 935-942, 1966.

Bower, G.H. A descriptive theory of memory. In: D.P. Kinkle (Ed.), Learning, Remembering, and Forgetting. Vol. 2 New York: New York Academy of Sciences, 1967, pp. 112-185(a).

Bower, G.H. A multicomponent theory of the memory trace. In: K. W. Spence and J. T. Spence (Eds.), The Psychology of Learning and Motivation: Advances in Research and Theory. Vol. 1. New York: Academic Press, 1967, pp. 229-235(b).

Broadbent, D.E. Perception and Communication. New York: Pergamon Press, 1958, pp. 297-301.

Broadbent, D.E. Communication models for memory. In: G.A. Talland and N.C. Waugh (Eds.), The Pathology of Memory. New York: Academic Press, 1969, pp. 167-171.

Broadbent, D.E. Recent analyses of short-term memory. In: K.H. Pribram and D.E. Broadbent (Eds.), Biology of Memory. New York: Academic Press, 1970, pp. 15-19.

Broadbent, D.E. Decision and stress. New York: Academic Press, 1971.

Carpenter, J.A. & Ross, B.M. Effect of alcohol on short-term memory. Quarterly Journal of Studies on Alcohol, 26: 561-579, 1965.

Cermak, L.S. & Butters, N. Information processing deficits of alcoholic Korsakoff patients. Quarterly Journal of Studies on Alcohol, 34: 1110-1132, 1973.

Craik, F.I.M. Two components in free recall. Journal of Verbal Learning and Verbal Behavior, 7: 996-1004, 1968.

Craik, F.I.M. The fate of primary memory items in free recall. Journal of Verbal Learning and Verbal Behavior, 9: 143-148, 1970.

Dallet, K.M. Number of categories and category information in free recall. Journal of Experimental Psychology, 68: 1-12, 1967.

Drachman, D.A. & Arbit, J.A. Memory and the hippocampal complex II. Is memory a multiple process? Archives of Neurology, 15: 52-61, 1966.

Frankel, F. & Cole, M. Measures of category clustering in free recall. Psychological Bulletin, 1971, 76, 39-44.

Glanzer, M. Distance between related words in free recall. Trace of the STS. Journal of Verbal Learning and Verbal Behavior, 8: 105-111, 1969.

Glanzer, M. Storage mechanisms in recall. In: G.H. Bower and J.T. Spence (Eds.), The Psychology of Learning and Motivation: Advances in Research and Theory. Vol. 5. New York: Academic Press, 1972, pp. 129-193.

Glanzer, M. & Cunitz, A.R. Two storage mechanisms in free recall. Journal of Verbal Learning and Verbal Behavior, 5: 351-360, 1966.

Glanzer, M., Gianutsos, R. & Dubin, S. The removal of items from short-term storage. Journal of Verbal Learning and Verbal Behavior, 8: 435-447, 1969.

Glanzer, M. & Schwartz, A. Mnemonic structure in free recall: Differential effects on STS and LTS. Journal of Verbal Learning and Verbal Behavior, 10: 194-198, 1971.

Huntley, M.S. Effects of alcohol, uncertainty, and novelty upon response selection. Psychopharmacologia, 39: 259-266, 1974.

Johnson, L.C. Sleep patterns in chronic alcoholics. In: N.K. Mello and J.H. Mendelson (Eds.), Recent Advances in Studies of Alcoholism. Washington,D.C. U.S. Government Printing Office, 1972, p. 288.

Kalin, R. Effects of alcohol on memory. Journal of Abnormal and Social Psychology, 69: 635-641, 1964.

Kintsch, W. Models for free recall and recognition. In: D.A. Norman (Ed.), Models of Human Memory. New York: Academic Press, 1970, pp. 333-374.

Laughery, K.R. Computer simulation of short-term memory: A component-decay model. In: J.T. Spence and G.H. Bower (Eds.), The Psychology of Learning and Motivation: Advances in Research and Theory. Vol. 3, New York: Academic Press, 1969, pp. 135-200.

Lester, B.K., Rundell, O.H., Cowden, L.C., & Williams, H.L. Chronic alcoholism, alcohol, and sleep. In: M. Gross (Ed.), Alcohol Intoxication and Withdrawal: Experimental Studies: Advances in Experimental Medicine and Biology. Vol. 35, New York: Plenum Press, 1973, pp. 261-279.

Lester, B.K., Rundell, O.H., Cowden, L.C., and Williams, H.L. Alcohol and sleep in the chronic alcoholic. In: N.R. Burch (Ed.), Proceedings of the Seventh Annual TRIMS Symposium. Houston, Texas, 1973 (in press)

Moskowitz, H. Laboratory studies of the effects of alcohol on some variables related to driving. Journal of Safety Research, 1973, 5, 185-199.

Moskowitz, H. & Roth, S. Effect of alcohol on response latency in object naming. Quarterly Journal of Studies on Alcohol, 32: 969-975, 1971.

Murdock, B.B., Jr. The serial position effect of free recall. Journal of Experimental Psychology, 64: 482-488, 1962.

Murdock, B.B., Jr. A fixed-point model for short-term memory. Journal of Mathematical Psychology, 4: 501-506, 1967 (b).

Murdock, B.B., Jr. Recent developments in short-term memory. British Journal of Psychology, 50: 421-433, 1967 (a).

Murdock, B.B., Jr. Short-term memory. In: G. H. Bower (Eds.), The Psychology of Learning and Motivation: Advances in Research and Theory. Vol. 5. New York: Academic Press, 1972, pp. 67-127.

Murdock, B.B. Jr. Human Memory: Theory and Data. New York: Academic Press, 1974.

Norman, D.A. Toward a theory of memory and attention. Psychological Review, 75: 522-536, 1968.

Norman, D.A. Memory and Attention: An Introduction to Human Information Processing. New York: Wiley, 1969.

Norman, D.A. & Rumelhart, D.E. A system for perception and memory. In: D.A. Norman (Ed.), Models of Human Memory. New York: Academic Press, 1970, pp. 19-64.

Oldfield, R.C. & Wingfield, A. Response latencies in naming objects. The Quarterly Journal of Experimental Psychology, 17: 273-281, 1965.
Phillips, J.L., Shiffrin, R.M. & Atkinson, R.C. Effects of list length on short-term memory. Journal of Verbal Learning and Verbal Behavior, 6: 303-311, 1967.
Posner, M.I. & Rossman, E. Effect of size and location of informational transforms upon short-term retention. Journal of Experimental Psychology, 70: 496-505, 1965.
Postman, L. & Phillips, L.W. Short-term temporal changes in free recall. Quarterly Journal of Experimental Psychology, 17: 132-138, 1965.
Puff, C.R. Clustering as a function of the sequential organization of stimulus word lists. Journal of Verbal Learning and Verbal Behavior, 5: 503-506, 1966.
Raymond, B. Short-term storage and long-term storage in free recall. Journal of Verbal Learning and Verbal Behavior, 8: 567-574, 1969.
Reitman, J.S. Mechanisms of forgetting in short-term memory. Cognitive Psychology, 2: 185-195, 1971.
Robinson, G., & Peebles, W. Interactions between alcohol, task difficulty, and compatibility in a choice reaction task. Perceptual and Motor Skills, 1974, 38, 459-466.
Rundell, O.H., Tharp, V., Lester, B.K. & Williams, H.L. Some effects of acute intoxication on information processing stages. Alcohol Technical Reports, 1: 25-32, 1973.
Shiffrin, R.M. Forgetting: Trace erosion or retrieval failure? Science, 168: 1601-1603, 1970.
Shiffrin, R.M. Information persistence in short-term memory. Journal of Experimental Psychology, 100: 39-49, 1973.
Shiffrin, R.M. & Atkinson, R.C. Storage and retrieval processes in long-term memory. Psychological Review, 76: 179-193, 1969.
Smith, E.E. Choice reaction time: An analysis of the major theoretical positions. Psychological Bulletin, 1968, 69, 77-110.
Sperling, G. The information available in brief visual presentations. Psychological Monographs, 74 (Whole No. 498), 1960.
Sperling, G. A model for human memory. Human Factors, 5: 19-31, 1963.
Sperling, G. Successive approximations to a model for short-term memory. Acta Psychologica, 27: 285-292, 1967.
Sternberg, S. The discovery of processing stages: Extensions of Donder's method. In: W. G. Koster (Ed.), Attention and performance. Acta Psychologica, 30: 276-315, 1969.
Storm, T. & Caird, W.K. The effects of alcohol on serial verbal learning in chronic alcoholics. Psychonomic Science, 9: 43-44, 1967.
Teichner, W.H. & Krebs, M.J. Laws of visual choice reaction time. Psychological Review, 81: 75-98, 1974.
Tharp, V.K., Jr., Rundell, O.H., Jr., Lester, B.K. & Williams, H.L. Alcohol and information processing. Psychopharmacologia, 40: 33-52, 1974.
Tulving, E. Subjective organization in free recall of "unrelated" words. Psychological Review, 69: 344-354, 1962.
Waugh, N.C. & Norman, D.A. Primary memory. Psychological Review, 72: 89-104, 1965.
Welford, A.T. Fundamentals of skill. London: Methuen, 1968.
Wickelgren, W.A. Multitrace strength theory. In: D.A. Norman (Ed.), Models of Human Memory. New York: Academic Press, 1970.